or risk. Ideally, it should provide an opportunity for intervention to prevent development of the disease, or at least result in better maternal and/or fetal outcomes [10].

Leptin (from the Greek "leptos"; meaning thin) is a protein hormone with important effects in regulating body weight, metabolism and reproductive functions [11]. The protein has 167 amino acid sequence containing one disulphide bond, it's molecular weight is about 16 KDs and has four helix bundle with one very short strand segment and two relative intermitting loops [12]. In pregnant women, leptin is synthesized in and secreted from placental trophoblast into maternal circulation at a considerable amount comparable with those in non pregnant woman [13]. Leptin is also produced by a culture of human choriocarcinoma cell line. Plasma leptin level is also markedly elevated in patient with Hydatidiform mole and choriocarcinoma, indicating that gestational trophoblastic neoplasms are leptin producing tumors. It has been demonstrated that placental production of leptin is augmented in women with severe preeclampsia [14].

Ouyang *et al*. [15] did a case control study between women with severe preeclampsia and normotensive women regarding serum leptin and found a significant elevation of serum leptin in women with severe preeclampsia. This finding pointed to the importance of leptin in the pathophysiology of preeclampsia and their involvement in the pathogenesis of the disease. As leptin causes oxidative stress in endothelial cells and has a calcifying effect on these cells, it has been suggested that leptin promote atherogenesis.

So, in pregnancy induced hypertension, placental ischemia is responsible for increased leptin level with increase in the inflammatory cytokines such as TNF alpha and IL-6 [16]. The aim of this work is to study the correlation between serum Leptin and severe preeclampsia.

Methods

This cross-sectional age-control study was done in the Department of Obstetrics and Gynecology/Baghdad Teaching Hospital-Medical City during the period from January 2010 to August 2010. A total number of 76 primipara in their third trimester where included in this study. Women with preexisting chronic hypertension, Diabetes mellitus, multiple pregnancies, chronic renal disease, chronic liver disease, and those with history of hyperuricemia were excluded from the study.

After taking detailed obstetrical and medical history 32 patients were having normal blood pressure without any history of prior hospitalization; while other 44 patients were presented with severe hypertension; diagnosed as systolic blood pressure of 160 mmHg and more and diastolic blood pressure of 110 mmHg and more, with a marked proteinuria on dipstick test in a random urine samples. After counseling and affordability of investigation, their blood samples were drawn for serum creatinine and uric acid.

Other samples were collected to obtain and clarify sera. Those samples were left to stand at room temperature for at least 30 minutes to allow the blood to clot, then centrifuged for 5 minutes, frozen at (-20 °C) and kept there without thawing till the day of testing. Then, serum leptin was measured using ELISA sandwich kits with the range of the assay from 0 to 100 ng/dl, also urine samples were taken for proteinuria by dipstick.

Results

Table 1a & b show that there was no statistically significant differences regarding body mass index between the two groups, while there were statistically significant differences regarding systolic and diastolic blood pressure ($P < 0.0001$). Their albumin in urine dipsticks on random urine samples show proteinuria in all cases of severe PET group (100%), while it was nil in all cases of the control group (100%).

There is statistically significant difference regarding serum leptin and serum uric acid between the two groups ($P = 0.0001$) While serum creatinine show mean±SD of 0.92 ± 0.18 in the severe PET group which is the upper